

DETAILED ACTION

Amendment after Non-final office action filed on April 23, 2009 is acknowledged. Claims 65-74 and 95-122 are pending in this application. On August 6, 2009, the Examiner called to propose Examiner's amendment to cancel claims 109-113 and 118. On August 24, 2009, Mr. Tener called back to indicate that the Examiner's amendment proposed was not accepted by the Applicant. An office action follows below.

1. Claims 65-74, 95-108 and 122 directed to an allowable product. Pursuant to the procedures set forth in MPEP § 821.04(B), claims 109-121, directed to the process of making or using an allowable product, previously withdrawn from consideration as a result of a restriction requirement, are hereby rejoined and fully examined for patentability under 37 CFR 1.104.

Because all claims previously withdrawn from consideration under 37 CFR 1.142 have been rejoined, **the restriction requirement as set forth in the Office action mailed on March 5, 2008 is hereby withdrawn.** In view of the withdrawal of the restriction requirement as to the rejoined inventions, applicant(s) are advised that if any claim presented in a continuation or divisional application is anticipated by, or includes all the limitations of, a claim that is allowable in the present application, such claim may be subject to provisional statutory and/or nonstatutory double patenting rejections over the claims of the instant application. Once the restriction requirement is withdrawn, the provisions of 35 U.S.C. 121 are no longer applicable. See *In re Ziegler*, 443 F.2d 1211, 1215, 170 USPQ 129, 131-32 (CCPA 1971). See also MPEP § 804.01.

Withdrawn Objections and Rejections

2. Objections to claims 70, 101-102 and 105 are hereby withdrawn in view of Applicant's amendment to the claims.
3. Rejections of claims 65-66, 70, 103-108 under 35 U.S.C. 112, second paragraph, as being indefinite, are hereby withdrawn in view of Applicant's amendment to the claims.
4. Rejection of claims 65-66, 70, 104-108 under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement, is hereby withdrawn in view of Applicant's amendment to the claims and persuasive arguments.
5. Rejection of claims 65-66, 70 and 104-108 under 35 U.S.C. 102(b) as being anticipated by Lee Moses NF (US Patent No. 5,273,991), is hereby withdrawn in view of Applicant's amendment and arguments.
6. Rejection of claims 65-66, 70 and 104-107 under 35 U.S.C. 102(b) as being anticipated by Lown et al (US Patent No. 4,912,199), is hereby withdrawn in view of Applicant's amendment and arguments.

New Rejection-35 U.S.C. 112, 1st

7. The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

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8. Claims 109-118 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the enablement requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to enable one skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention.

The factors to be considered in determining whether a disclosure meets the enablement requirement of 35 U.S.C. 112, first paragraph, have been described in In re Wands, 8 USPQ2d 1400 (Fed. Cir. 1988). Among these factors are: (1) the nature or the invention; (2) the state of the prior art; (3) the relative skill of those in the art; (4) the predictability or unpredictability of the art; (5) the breadth of the claims; (6) the amount of direction or guidance presented; (7) the presence or absence of working examples; and (8) the quantity of experimentation necessary. When the above factors are weighed, it is the examiner's position that one skilled in the art could not practice the invention without undue experimentation.

(1) The nature of the invention and (5) The breadth of the claims:

The claims are drawn to a method of treatment of a disease that relies upon DNA replication for its propagation, a method of treatment of cancer, and a method of inhibiting DNA replication, comprising administering a therapeutically effective amount of a compound of formula I. Claims 114-117 are drawn to a combination product comprising components (A) a formulation comprising a compound of formula I, and (B)

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a formulation comprising one or more other chemical agents that are known to be effective in treating diseases that rely upon DNA replication for their propagation.

(2) The state of the prior art:

In regards to treatment of a disease that relies upon DNA replication for its propagation, a disease that relies upon DNA replication can be any disease. These include any viral diseases, such as HIV-1, HCV, H1N1, common cold, Herpes, cancer, and any other diseases related to DNA replication.

The Merck manual indicates that HIV-1 infection results from 1 of 2 similar retroviruses (HIV-1 and HIV-2) that destroy CD4+ lymphocytes and impair cell-mediated immunity, increasing the risk of certain infection and cancers (see Merck manual, HIV-1 Introduction, 1st paragraph). The Merck manual indicates that retroviruses are enveloped RNA viruses defined by their mechanism of replication via reverse transcription to produce DNA copies that integrate in the host cell genome (see Merck manual, HIV-1 Introduction, 3rd paragraph). The Merck manual indicates that viral replication requires that reverse transcriptase (an RNA-dependent DNA polymerase) copy HIV RNA, producing proviral DNA, and this copying is prone to errors, resulting in frequent mutations. And these mutations facilitate the generation of HIV that can resist control by the host's immune system and by antiretroviral drugs (see Merck manual, HIV-1 Introduction, Pathophysiology). Further, the Merck manual indicates that the high volume of HIV replication and high frequency of transcription errors by HIV reverse transcriptase results in many mutations, increasing the chance of producing strains to

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host immunity and drugs (see Merck manual, HIV-1 Introduction, Pathophysiology). The Merck manual indicates that primary HIV infection may be asymptomatic or cause transient nonspecific symptoms (see Merck manual, HIV-1 Introduction, Symptoms and Signs). Diagnosis is by HIV antibody testing and nucleic acid amplification assays to determine HIV RNA level (see Merck manual, HIV-1 Introduction, Diagnosis). The Merck manual indicates that highly active antiretroviral therapy (HAART) aims to suppress viral replication to undetectable levels. Partial suppression (failure to lower plasma levels to undetectable levels) may select for single or multiple mutations in HIV that make viruses completely or partially resistant and make subsequent treatment more likely to fail (see Merck manual, HIV-1 Introduction, Treatment). Furthermore, the Merck manual indicates that combinations of 3 or 4 drugs from different classes are usually necessary to fully suppress replication of wild-type HIV. Interactions between antiretrovirals may decrease the efficacy of each drug. Further, combining drugs often increase the risk that either drug will have an adverse effect (see Merck manual, HIV-1 Introduction, Treatment). The Merck manual indicates that vaccines against HIV have been difficult to develop because HIV surface proteins mutate easily, resulting in an enormous diversity of antigenic types (see Merck manual, HIV-1 Introduction, Prevention).

In regards to Respiratory viruses, the Merck manual indicates that viral infections commonly affect the upper or lower respiratory tract. The severity of viral respiratory illness varies widely, and morbidity may result directly from viral infection or may be indirect (see Merck manual, Respiratory viruses, Introduction). The Merck manual

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indicates that antibacterial drugs are ineffective against viral pathogens, and prophylaxis against secondary bacterial infection is not recommended. The Merck manual indicates that a guanosine analog that inhibits replication of many RNA and DNA viruses, may be considered in severely immunocompromised patients with lower respiratory tract infection due to RSV (see Merck manual, Respiratory viruses, Introduction, Treatment).

The Merck manual indicates that Severe Acute Respiratory Syndrome (SARS) is caused by a coronavirus, and has an incubation of 2 to 10 days (see Merck manual, SARS, 1st paragraph). The Merck manual indicates that initial symptoms of SARS resemble influenza, with fever, cough, chills, rigor, and myalgia. Death is due to respiratory failure. The Merck manual indicates that because initial symptoms are nonspecific, SARS is suspected in patients with likely exposure as well as fever and suggestive clinical symptoms (see Merck manual, SARS, Symptoms, Signs, and Diagnosis). The Merck manual indicates that treatment of SARS is supportive, with mechanical ventilation as needed. Such drugs as oseltamivir, ribavirin, and corticosteroids have been used, but there is not current evidence of benefit (see Merck manual, SARS, Prognosis, Treatment, and Prevention).

The Merck manual indicates that acute viral hepatitis is diffuse liver inflammation caused by specific hepatotropic viruses that have diverse modes of transmission and epidemiologies (see Merck manual, Acute Viral Hepatitis). The Merck manual indicates that there are 5 different types of hepatitis viruses (A-E) (see Merck manual, Acute Viral Hepatitis, Table 2). For example, the Merck manual indicates that HCV is a single-stranded RNA flavivirus, and six major HCV subtypes exist with varying amino acid

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sequences. HCV can also alter its amino acid pattern over time in an infected person (see Merck manual, Acute Viral Hepatitis, Etiology and Epidemiology). The Merck manual indicates that no treatment attenuate acute viral hepatitis except, postexposure immunoprophylaxis (see Merck manual, Acute Viral Hepatitis, Treatment).

Mosch et al (The Journal of Neuroscience, 2007, 27(26): 6859-6867) indicates that reactivation of the cell cycle, including DNA replication, might play a major role in Alzheimer's disease (see abstract). The reference teaches that the pattern of disease progression matches the regional degree of neuronal plasticity and inversely recapitulates ontogenetic and phylogenetic brain development (see p. 6859, left column, Introduction). The reference further indicates that the expression of developmentally regulated genes in AD that correspond to a condition of dedifferentiation and links neurodegeneration to cell-cycle-related events (see p.6859, right column). Furthermore, the Merck manual indicates that Alzheimer's disease is chronic, global, usually irreversible deterioration of cognition. The main types of Alzheimer's disease are: vascular dementia, Lewy body dementia, frontal-temporal dementias, and HIV-associated dementia (See Merck manual, "Dementia", Etiology and Classification, 2nd paragraph). Furthermore, Alzheimer's disease causes progressive cognitive deterioration and is characterized by senile plaques, beta-amyloid deposits, and neurofibrillary tangles in the cerebral cortex and subcortical gray matter (see Merck manual in Dementia under "Alzheimer's disease). Furthermore, the Merck manual indicates that most cases are sporadic, with late onset and unclear etiology (see Merck manual, "Alzheimer's disease", Etiology and Pathophysiology). Since symptoms, signs

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are similar to those of other dementia, distinguishing Alzheimer's disease from other dementias is difficult (see Merck Manual, "Alzheimer's disease", Symptoms, Signs, and Diagnosis). Furthermore, Mattson MP (Nature, 2004, 430: 631-639) indicates that the risk of Alzheimer's disease (AD) dramatically increases in individuals beyond the age of 70 (see p. 631, left column, 1st sentence). The vast majority of cases of AD are sporadic, they do not run in families...molecular genetic analyses suggest that there are likely many genes that influence one's susceptibility to AD (see p. 633, left column, 2nd paragraph). Additionally, Mattson indicates that although drugs can temporarily improve memory, at present there are no treatments that can stop or reverse the inexorable neurodegenerative process (see abstract).

In regards to "treating cancers", Merck manual indicates that cancer is an unregulated proliferation of cells due to loss of normal controls, resulting in unregulated growth, lack of differentiation, local tissue invasion, and often, metastasis. Cancer can develop in any tissue or organ at any age. Furthermore, the Merck manual indicates that many cancers are curable if detected at an early stage, and long-term remission is often possible in later stages. However, cure is not always possible and is not attempted in some advanced cases in which palliative care provides better quality of life than vigorous but fruitless attempts at tumor eradication (see Merck manual, Introduction). Additionally, the Merck manual indicates that malignancy may lead to pain, wasting, neuropathy, nausea, anorexia, seizures, hypercalcemia, hyperuricemia, or obstruction...Death typically occurs as a result of sudden or progressive failure of one or multiple organ systems (see Merck manual, Clinical Aspects of Cancer).

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Furthermore, a complete history and physical examination may reveal unexpected clues early cancer (see Merck manual, Clinical Aspects of Cancer, Diagnosis).

Furthermore, arts indicate the difficulties in going from *in vitro* to *in vivo* for drug development for treatment of cancers. Auerbach et al (Cancer and Metastasis Reviews, 2000, 19: 167-172) indicates that one of the major problems in angiogenesis research has been the difficulty of finding suitable methods for assessing the angiogenic response. For example, the 96 well rapid screening assay for cytokinesis was developed in order to permit screening of hybridoma supernatants...*In vitro* tests in general have been limited by the availability of suitable sources for endothelial cells, while *in vivo* assays have proven difficult to quantitate, limited in feasibility, and the test sites are not typical of the *in vivo* reality (see p. 167, left column, 1st paragraph). Gura T (Science, 1997, 278(5340): 1041-1042, encloses 1-5) indicates that “the fundamental problem in drug discovery for cancer is that the model systems are not predictive at all” (see p. 1, 2nd paragraph). Furthermore, Gura T indicates that the results of xenograft screening turned out to be not much better than those obtained with the original models, mainly because the xenograft tumors don’t behave like naturally occurring tumors in humans—they don’t spread to other tissues, for example (see p. 2, 4th paragraph). Further, when patient’s tumor cells in Petri dishes or culture flasks and monitor the cells’ responses to various anticancer treatments, they don’t work because the cells simply fail to divide in culture, and the results cannot tell a researcher how anticancer drugs will act in the body (see p. 3, 7th paragraph). Furthermore, Jain RK (Scientific American, July 1994,58-65) indicates that the existing pharmacopoeia has not markedly reduced

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the number of deaths caused by the most common solid tumors in adults, among them cancers of the lung, breast, colon, rectum, prostate and brain (see p. 58, left most column, 1st paragraph). Further, Jain RK indicates that to eradicate tumors, the therapeutic agents must then disperse throughout the growths in concentrations high enough to eliminate every deadly cells...solid cancers frequently impose formidable barriers to such dispersion (see p. 58, bottom of the left most column continuing onto the top of the middle column). Jain RK indicates that there are 3 critical tasks that drugs must do to attack malignant cells in a tumor: 1) it has to make its way into a microscopic blood vessel lying near malignant cells in the tumor, 2) exit from the vessel into the surrounding matrix, and 3) migrate through the matrix to the cells. Unfortunately, tumors often develop in ways that hinder each of these steps (see p. 58, bottom of right most column). Thus, the art recognizes that going from *in vitro* studies to *in vivo* studies for cancer drug developments are difficult to achieve.

The art indicates that the disease must be determined first to determine the type of treatment and the effective amount to be administered. The art provide guidance as to how to treat cancers, certain viral infections, and temporarily improve memory (in AD). However, the art also indicates the difficulties in going from *in vitro* to *in vivo* for drug development for treatment of cancers.

(3) *The relative skill of those in the art:*

The relative skill of those in the art is high.

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(4) The predictability or unpredictability of the art:

The arts indicate that there is unpredictability in treating diseases that relies upon DNA replication. For example, as indicated above, due to the mutation of HIV-1 virus, drug cocktails should be administered to the patient population. However, due to the drug interactions between antiretrovirals may decrease the efficacy of each drug. Even Alzheimer's disease indicated in DNA replication. The claims do not identify the patient population, therefore, the claims imply that anyone can be treated for any and all diseases that rely upon DNA replication. However, there are different mechanisms, different cells and different symptoms involved in diseases that rely upon DNA replication. There are too many variables between the experimentation, thus, it clearly shows the unpredictability of the art.

(6) The amount of direction or guidance presented and (7) The presence or absence of working examples:

The specification discloses the method of making the compounds of formula I (see Examples 1-33). Furthermore, the specification describes methods to determine the binding of the compounds to the minor groove of DNA by the capillary electrophoresis and/or DNA footprinting (see Examples 34-35). Example 36 describes the enhancement of DNA duplex and stabilization of DNA duplex formation by increase in melting temperature (T_m). The working Example 37 describes inhibition of the growth of microorganisms by measuring the minimum inhibition concentration (MIC) versus the antibiotic controls.

The specification does not disclose how to treat any and all diseases that rely upon DNA replication. The specification does not disclose any *in vitro* or *in vivo* data on cancer treatment. Even the inhibition of growth of microorganisms disclosed in the specification shows unpredictability. For example, Table A shows that compounds 7, 17, 28 and 3 did not work on *E. coli* as compared to the amoxicillin control. For *S. aureus*, compound 28 worked poorly. Table B shows that compounds 7, 28 and 3 inhibited *S. faecalis*, but not as well as the amoxicillin control. Compound 17 worked for *P. vulgaris*, but not as well as the amoxicillin control; Compounds 7, 28 and 3 inhibited poorly. The working example 37 indicates that depending on different viruses and different diseases, it is unpredictable as to which compounds would work and which compounds would not. Since some of the compounds were ineffective against certain virus, fungus and bacteria, it is unpredictable which compounds would work to treat all viral, bacterial, fungal or other microbial infections. Additionally, it is unclear as to when the compound is to be administered and the patient population. The working examples are directed towards inhibition of growth of microorganisms by measuring the MIC on different viruses, bacteria and fungus. Furthermore, the specification does not disclose what DNA sequences are to be inhibited. The specification does not disclose whether or not these DNA that are to be inhibited enters the cells. Additionally, the specification does not define the specificity of DNA sequences that are to be inhibited. For example, there are vast numbers of DNA in a patient being administered the compound. If the compound is to inhibit all and every DNA, then these compounds would also inhibit

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essential endogenous DNA. Therefore, these would destroy the essential DNA that is needed for survival of the patient.

As described supra, a disease that relies upon DNA replication can be any disease. These include any viral diseases, such as HIV-1, HCV, H1N1, common cold, Herpes, cancer, and any other diseases related to DNA replication.

The Merck manual indicates that HIV-1 infection results from 1 of 2 similar retroviruses (HIV-1 and HIV-2) that destroy CD4+ lymphocytes and impair cell-mediated immunity, increasing the risk of certain infection and cancers (see Merck manual, HIV-1 Introduction, 1st paragraph). The Merck manual indicates that retroviruses are enveloped RNA viruses defined by their mechanism of replication via reverse transcription to produce DNA copies that integrate in the host cell genome (see Merck manual, HIV-1 Introduction, 3rd paragraph). The Merck manual indicates that viral replication requires that reverse transcriptase (an RNA-dependent DNA polymerase) copy HIV RNA, producing proviral DNA, and this copying is prone to errors, resulting in frequent mutations. And these mutations facilitate the generation of HIV that can resist control by the host's immune system and by antiretroviral drugs (see Merck manual, HIV-1 Introduction, Pathophysiology). Further, the Merck manual indicates that the high volume of HIV replication and high frequency of transcription errors by HIV reverse transcriptase results in many mutations, increasing the chance of producing strains to host immunity and drugs (see Merck manual, HIV-1 Introduction, Pathophysiology). The Merck manual indicates that primary HIV infection may be asymptomatic or cause transient nonspecific symptoms (see Merck manual, HIV-1 Introduction, Symptoms and

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Signs). Diagnosis is by HIV antibody testing and nucleic acid amplification assays to determine HIV RNA level (see Merck manual, HIV-1 Introduction, Diagnosis). The Merck manual indicates that highly active antiretroviral therapy (HAART) aims to suppress viral replication to undetectable levels. Partial suppression (failure to lower plasma levels to undetectable levels) may select for single or multiple mutations in HIV that make viruses completely or partially resistant and make subsequent treatment more likely to fail (see Merck manual, HIV-1 Introduction, Treatment). Furthermore, the Merck manual indicates that combinations of 3 or 4 drugs from different classes are usually necessary to fully suppress replication of wild-type HIV. Interactions between antiretrovirals may decrease the efficacy of each drug. Further, combining drugs often increase the risk that either drug will have an adverse effect (see Merck manual, HIV-1 Introduction, Treatment). The Merck manual indicates that vaccines against HIV have been difficult to develop because HIV surface proteins mutate easily, resulting in an enormous diversity of antigenic types (see Merck manual, HIV-1 Introduction, Prevention).

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considered in severely immunocompromised patients with lower respiratory tract infection due to RSV (see Merck manual, Respiratory viruses, Introduction, Treatment).

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Diagnosis). Furthermore, Mattson MP (Nature, 2004, 430: 631-639) indicates that the risk of Alzheimer's disease (AD) dramatically increases in individuals beyond the age of 70 (see p. 631, left column, 1st sentence). The vast majority of cases of AD are sporadic, they do not run in families...molecular genetic analyses suggest that there are likely many genes that influence one's susceptibility to AD (see p. 633, left column, 2nd paragraph). Additionally, Mattson indicates that although drugs can temporarily improve memory, at present there are no treatments that can stop or reverse the inexorable neurodegenerative process (see abstract).

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Furthermore, arts indicate the difficulties in going from *in vitro* to *in vivo* for drug development for treatment of cancers. Auerbach et al (Cancer and Metastasis Reviews, 2000, 19: 167-172) indicates that one of the major problems in angiogenesis research has been the difficulty of finding suitable methods for assessing the angiogenic response. For example, the 96 well rapid screening assay for cytokinesis was developed in order to permit screening of hybridoma supernatants...*In vitro* tests in general have been limited by the availability of suitable sources for endothelial cells, while *in vivo* assays have proven difficult to quantitate, limited in feasibility, and the test sites are not typical of the *in vivo* reality (see p. 167, left column, 1st paragraph). Gura T (Science, 1997, 278(5340): 1041-1042, encloses 1-5) indicates that “the fundamental problem in drug discovery for cancer is that the model systems are not predictive at all” (see p. 1, 2nd paragraph). Furthermore, Gura T indicates that the results of xenograft screening turned out to be not much better than those obtained with the original models, mainly because the xenograft tumors don’t behave like naturally occurring tumors in humans—they don’t spread to other tissues, for example (see p. 2, 4th paragraph). Further, when patient’s tumor cells in Petri dishes or culture flasks and monitor the cells’ responses to various anticancer treatments, they don’t work because the cells simply fail to divide in culture, and the results cannot tell a researcher how anticancer drugs will act in the body (see p. 3, 7th paragraph). Furthermore, Jain RK (Scientific American, July 1994, 58-65) indicates that the existing pharmacopoeia has not markedly reduced the number of deaths caused by the most common solid tumors in adults, among them cancers of the lung, breast, colon, rectum, prostate and brain (see p. 58, left most column, 1st

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paragraph). Further, Jain RK indicates that to eradicate tumors, the therapeutic agents must then disperse throughout the growths in concentrations high enough to eliminate every deadly cells....solid cancers frequently impose formidable barriers to such dispersion (see p. 58, bottom of the left most column continuing onto the top of the middle column). Jain RK indicates that there are 3 critical tasks that drugs must do to attack malignant cells in a tumor: 1) it has to make its way into a microscopic blood vessel lying near malignant cells in the tumor, 2) exit from the vessel into the surrounding matrix, and 3) migrate through the matrix to the cells. Unfortunately, tumors often develop in ways that hinder each of these steps (see p. 58, bottom of right most column). Thus, the art recognizes that going from *in vitro* studies to *in vivo* studies for cancer drug developments are difficult to achieve.

There is no clear guidance as to how to determine the patient population, who would have a disease that relies upon DNA replication, and the effective amount to treat all and any disease, and cancer. For example, cancer is an unregulated proliferation of cells due to loss of normal controls, resulting in unregulated growth, lack of differentiation, local tissue invasion, and often, metastasis. Cancer can develop in any tissue or organ at any age, and it is unclear who would develop cancers and other viral infections, for example HIV-1, HCV and common cold, more guidance is necessary.

(8) The quantity of experimentation necessary:

There are plethora of diseases that involve DNA replication. Furthermore, there are different mechanisms, cells and symptoms involved with these diseases. Since it is

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uncertain to predict the patient population who are susceptible to any and all diseases that rely upon DNA replication, including viral infections (HIV-1, HCV, H1N1, common cold and so on), cancer and Alzheimer's disease, the art indicates the unpredictability of different types of diseases, and the Applicant's data show unpredictability of compounds on different types of infections, one of ordinary skill in the art would be burdened with undue "painstaking experimentation study" to determine if the compounds of formula I would be effective in treating diseases that rely upon DNA replication, treating viral, bacterial, fungal or other microbial infection, and inhibiting all DNA replication.

Conclusion

9. Claims 65-74, 95-108, 119-122 appear to be allowable. Claims 109-118 are rejected.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of

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the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to JULIE HA whose telephone number is (571)272-5982.

The examiner can normally be reached on Mon-Thurs, 5:30 AM to 4:00 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Cecilia Tsang can be reached on 571-272-0562. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Julie Ha/
Examiner, Art Unit 1654

/Cecilia Tsang/
Supervisory Patent Examiner, Art Unit 1654

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